PUBLIC HEALTH REPORTS

VOL. 53 FEBRUARY 11, 1938 NO. 6

HARMFUL INDUSTRIAL DUSTS[1]

By R. R. SAYERS, *Senior Surgeon, United States Public Health Service*[2]

While dusts exist everywhere in the atmosphere, it has been recognized for centuries that workers in certain dusty occupations are less healthy than those not so exposed. The number of persons exposed in dusty occupations comprises one of the largest industrial groups. The occupational diseases of workers in dusty atmospheres have been found to be due to entrance of dust into the system by inhalation, by ingestion, by direct absorption through the skin, by irritation of the skin, or by a combination of the foregoing.

The suspensions of particulate matter in air have been broadly termed dusts, fumes, and smokes. Such a classification is necessarily arbitrary, since the line of demarcation between them is not very sharp, and the chief differentiation is based on the size of the particles. Investigations by the Public Health Service in dusty industries (*1*) revealed that about 70 percent of the dust particles examined were between 1 and 3 microns in size, only about 20 percent were less than 1 micron, and the median size was 1.3 microns. Industrial dusts are mainly less than 10 microns in size. However, fibrous dusts in the air, such as asbestos, have been found to contain particles as large as 200–400 microns in the greatest diameter.

While we cannot agree with Collis that, generally speaking, dusts are more injurious as their chemical composition differs from that of the human body, or from the elements of which the body is normally composed (*2*), it is difficult to establish a comprehensive definition for *toxic* dusts. Sollmann (*3*), after analysis of various definitions for poison, felt that it was very difficult to give a definition which would not be ambiguous in some cases, but believed that the following covered most of the points which must be considered in classifying a substance as such:

> A poison is any substance which, acting directly through its inherent chemic properties, and by its ordinary action, is capable of destroying life, or of seriously endangering health, when it is applied to the body, externally, or in moderate doses (to 50 gm) internally.

Some dusts are known to be poisonous, while others, in the concentrations usually encountered, are comparatively harmless. How-

[1] Presented before the National Safety Congress, Kansas City, Mo., Oct. 13, 1937.

[2] Division of Industrial Hygiene of the National Institute of Health.

ever, it may be stated that *breathing* dust in high concentration is not desirable.

According to Fairhall, the damage arising from the inhalation of toxic dust may be either local or remote, and depends upon whether the material is a protoplasmic poison, whether it is caustic in reaction, or whether it is absorbed into the blood stream and carried to other centers which are in turn affected (*4*).

It is now well established that exposure to certain kinds of dusts, such as those containing considerable quantities of free silica, has increased the morbidity and mortality rate from respiratory disease; while metallic dusts, such as lead and its compounds, have been associated with general systemic poisoning (*5*). Dust may be swallowed with saliva, water, or food, and direct poisoning has been traced to absorption by this method; other dusts may act as irritants and produce affections of the skin, irritate mucous membranes of nose, eyes, and throat, often causing inflammatory diseases of these organs (*6*).

Some inorganic dusts are able to penetrate the deep lung tissues and are sufficiently insoluble to be retained there. Some of these may be active in the human tissues, causing definite and permanent injury, while others are inert and may be retained for years, apparently without serious damage; some are gradually absorbed without causing pathological changes (*7*).

Most inhaled particles are soluble, at least to a small extent. If harmless, cell activity is stimulated so that phagocytes remove the dust. If the solute is toxic, the viability of the phagocyte is affected and an ineffective accumulation results. At the same time, further solute may diffuse into neighboring tissues, setting up an irritation and subsequent fibrosis. The nature as well as the solubility of the solute is an important factor, but of two substances of approximately equal toxicity, the more soluble form causes the greater damage. However, substances of such low solubility as silica may ultimately produce extensive injury (*4*).

Kettle has stated that harmful dusts, if inhaled into the lungs, may activate a latent tuberculous infection; they may exaggerate an active tuberculous lesion or a coincident infection. If a harmless dust is inhaled in sufficient quantities, some of it remains in the lungs and causes a mild degree of fibrosis merely through mechanical irritation, but such fibrosis is never sufficient to interfere with the function of the lung (*8*).

Some writers state that while the influence of nonpoisonous dusts on health is a debatable subject, abnormally high death rates from bronchitis and pneumonia are sometimes attributed to chronic exposure to nontoxic dusts. Large amounts of dust are occasionally found in supposedly normal lungs at autopsy (*9*).

According to Drinker, there are four different types of reaction produced in man by the inhalation of dust. The first and most important are the pneumoconioses, such as silicosis and asbestosis, which cause specific lung pathology and often are followed by pulmonary tuberculosis. The second type of reaction is caused by toxic dusts, like lead, cadmium, and radium. A third type of malady follows the inhalation of finely divided metallic fume particles, such as zinc oxide, and is known as metal fume fever. The fourth reaction, allergic in character, is caused by breathing organic dusts, such as pollen and certain types of pulverized wood and flour. In all four cases the sole cause of the disability may be dust inhalation, but the reactions from toxic dusts result from swallowing as well as from inhalation (*10*).

For various reasons it is difficult to set up an absolute classification for dusts. Opinion is rapidly changing regarding so-called inert or harmless dusts and further investigation may prove some of them to be injurious. Some dusts may have both a toxic and an irritant action; while, on the other hand, the poisoning resulting from exposure to a dust may be the combined effect of more than one mode of entrance into the body. However, the following classification of dusts, according to physical characteristics and physiological effect, is used for convenience.

I. ORGANIC DUSTS

Organic dusts are those which contain carbon, and were originally supposed to come from organized substances derived from animal or plant life. Living dusts come under this classification and are thought to be of the same order of size as industrial dusts, with which they are frequently associated. Bacteria are usually between 0.5 and 3 microns, except in a few cases, such as the anthrax bacillus, which ranges from 1 to 1.25 micron in breadth and 4.5 to 10 microns in length. Spores of fungi may range in size from less than 1 micron to 20 microns in length and up to 12 microns in diameter. However, it is possible that the larger and heavier varieties, like the larger and heavier dust particles, settle by gravity, and do not remain suspended in the air.

Many thousands of organic substances, carbon-containing, are made synthetically by chemical processes, such as dyestuffs, explosives, and similar substances.

1. NONLIVING ORGANIC DUSTS

As the name implies, these are composed of nonviable particles, which may or may not be inherently toxic or irritant, but which nevertheless produce untoward effects in the human organism. "Allergic" dusts, or those to which only certain persons are or may become hypersensitive, with resulting asthma, rhinitis, or other disturbances, are included in this classification.

(*a*) *Toxic and* (*or*) *irritant dusts.*—Toxic or irritant dusts are all organic dusts which produce untoward symptoms, either systemic or local. Those producing local symptoms are usually described as irritant; those producing general or systemic symptoms are termed toxic. A dust may be both toxic and irritant.

In reported cases of injury or death following inhalation of dust from organic compounds, among the chief offenders are paranitraniline, the dinitrobenzenes, chlorodinitrobenzenes, trinitrophenol, and nitronaphthalene. Eye damage due to nflammation of the cornea has occurred among workers exposed to methyl violet dust. The dust from paraphenylene diamine derivatives is particularly irritating and dangerous, causing not only a severe form of dermatitis where the dust comes in contact with the skin, but also producing acute inflammation of the mucous membrane of the respiratory passages (*4*). Toxic dusts may be generated during the handling of powdered dyes and in preliminary dyeing operations, particularly the hydro-extractor process, where particles are disseminated in all directions (*11*). Coal tar and indigo dyes are substances most frequently used.

Cases of amblyopia have been reported from exposure to inhalation of tobacco dust (*12*). It has been found that inhaled tobacco dust exerts a nicotine action on the organism 15 times greater than that produced by the same quantity of smoked tobacco with an equal nicotine content (*13*).

Severe dermatitis is experienced by many workers handling powder containing TNT, which is used in making high explosives. Picric acid, which is also used for this purpose, requires drying, and in this latter state it has been found to produce a dermatitis. Picric acid is also the oldest synthetic organic dyestuff (*14*). Dermatitis occurs among handlers of silk with varying frequency. Salls determined that a rash occurring among workers in a silk factory was due to dust from silk cocoons imported from Africa (*15*).

Persons engaged in the manufacture of quinine and quinine preparations suffer from skin phenomena, which occur for the most part on exposed parts of the body, and may be caused by ingestion or by quinine dust, or powder form of the preparation, exerting a direct irritating effect on the skin (*16*). Dermatitis due to other vegetable dusts such as vanilla, powdered arnica, pyrethrum, etc., has been reported (*14*).

Dental lesions, of occupational origin, have been reported among workers in sugar. The gingivitis caused by sugar dust is classified as purely mechanical, with later-developing caries. Digestive disorders, respiratory conditions, and cutaneous conditions (especially of the face) have also been reported (*17*).

Dock workers, transporters of grain, workers in grain and flour mills, and persons in similar occupations are exposed to dusts during

their work. Dusts from cereals may contain many impurities, which may cause an irritating action on the respiratory passages, as well as inflammation of the skin. Digestive disturbances, dental defects, diminution in hearing, and conjunctivitis have been observed among millers, and have been attributed to the dusts to which such workers are exposed (*18*).

(*b*) *Allergic.*—Many apparently innocuous substances may produce reactions in persons of peculiar personal susceptibility. The term "allergy" is used to describe this condition of hypersensitiveness, or susceptibility, and allergic phenomena most frequently manifest themselves in skin reactions. However, they may cause acute reactions elsewhere in the body. When the respiratory tract is involved we have such well-known diseases as hay fever or asthma. These diseases may develop as a result of hypersensitiveness to such substances as pollens from plants, horsehair, furs, feathers, and the like. For instance, furriers, workers in clothing industries who are exposed to wool dust, and others may suffer from hay fever or asthma (*19*). It is unnecessary for the offending dust to reach the depths of the lungs (giant pollens, for example, are reported to produce their effect after being caught in the upper respiratory passages). Should such an offending substance be finely ground, it could reach the alveoli and as a result probably all physiologic reactions would be accelerated (*20*).

In a survey of a plant where resin is mixed, ground, and molded, it was found that 80 percent of the occupational dermatitis there was due to hypersensitivity to hexamethylenetetramine and formaldehyde contained in the dust to which the workers were exposed (*21*). During the first processes of cotton spinning, cotton-strippers are exposed to dust arising from cotton husks and debris, which produces a typical form of asthma (*2*).

Ordinary wood dust has been of interest, owing to its purely mechanical action; but still greater care is required in handling certain kinds, especially woods coming from abroad, because of the essential oils impregnating them, which when freed in the dust may affect the health of the workers concerned. Some of the woods capable of causing skin lesions are Brazil wood, satinwood, teakwood, cumaru or tonka wood, black ebony wood, West Indian mahogany, Japanese tagayasan, coccoloba, chestnutwood, olivewood, and California sequoia (redwood). All persons who handle these woods are not injured, only those particularly susceptible to the substances they contain becoming affected (*22*).

2. LIVING ORGANIC DUSTS

Living organic dusts contain particles capable of exhibiting the phenomena of life (*2*) (especially the property of reproduction or multiplication), such as bacteria and fungi. They are usually found

in low concentrations and are associated with nonliving dusts in the air.

(*a*) *Bacteria.*—One of the most important among these is the anthrax bacillus, which is contained in the dust from skins, furs, wool, and animal hair, horns, hoofs, bones, and similar animal products. This disease may occur in two forms; namely, *cutaneous* (in which the organism affects the skin) and *pulmonary* (when it is inhaled, as in the form of anthrax known as wool-sorters' disease) (*12*).

Cases of tetanus reported in connection with jute manufacture were traced to the raw material, the bacillus having been found in the factory dust (*23*). Diphtheria, tuberculosis, smallpox, typhoid, and other bacillus-produced diseases, may result from exposure to infected dusts.

Bacterial sensitization can be the cause of any of the allergic diseases; namely, asthma, perennial hay fever, urticaria, angioneurotic edema, eczema, or migraine headaches (*24*).

(*b*) *Fungi.*—Dusts containing the mycelia and spores of parasitic fungi give rise to annoyance and discomfort. "Maltster's" itch from the dust alone has been reported. In Provence, reeds used for ceilings are stacked while still wet and undergo fermentation; they become covered with a white powder (a dry fungus of the Mucor family, *Sporotrichum dermatodes*), which is scattered when the bark is stripped off. This powder is irritating to the skin and mucous membranes. Mycelia and spores of molds are commonly found to cause rashes; an example is the black powder coming from macerated sugarcane stalks. Among basket makers, the mycelium and spores, in the form of a white mold (hyphomycete) from the rattan canes used, get shaken out when the canes are split, hammered, and cut, causing painful fissures to develop on the skin where they alight (*14*).

A form of asthma or spasmodic cough, suffered by cotton weavers and known as aspergillosis, has been considered due to inhalation of spores of a mildew which sometimes occurs on the threads (*23*). In a study of silicosis among miners, made by the Public Health Service, a number of cases of typical miliary calcification were encountered. Unstained smears from those cases examined were positive for fungus, two types of *Aspergillus* fungi being identified. All of the subjects but one were farmers, teamsters, feedmill workers, or residents of small agricultural towns where grain is marketed. Farmers are exposed to fungi in threshing wheat, baling hay, or handling various small grains (*25*).

Some other fungus diseases, such as actinomycosis and blastomycosis, are associated with occupational exposure to dust. The former occurs among workers handling straw, hay, grass, vegetable debris contaminated with mold, and similar material. Actinomycosis is likely to affect people engaged in commercial handling, storing, and

cleansing of grain (grain distilleries, flour mills, grain crushing, breweries, and malting houses, etc.) (*26*).

II. INORGANIC DUSTS

Inorganic compounds are of mineral origin, not requiring a living organism to produce them (*27*). A number of dusts not usually classed as toxic may, under some conditions, produce untoward effects on the human organism. Classified under inorganic are toxic and (or) irritant, fibrosis-producing and non-fibrosis-producing dusts.

(*a*) *Toxic and* (*or*) *irritant.*—Toxic dusts are those which are inherently toxic when inhaled, ingested, or otherwise absorbed. Among those which produce systemic poisoning, some of which are also irritant, are the dusts from heavy metals and their salts, such as lead, mercury, arsenic, cadmium, zinc, and similar metals. Irritant dusts are injurious by reason of their strong irritative or corrosive properties. As a rule, inhaled irritant substances immediately cause a reaction in the upper respiratory tract of such severity that they are prevented from reaching the lungs, although they may cause lung damage by extension of inflammation if the mucous membrane is corroded (*7*). Lime, calcium oxide, and the dichromates are examples of irritant dusts. An inorganic dust may possess both toxic and irritant properties, and the poisoning produced may be the combined effect of more than one mode of entrance into the body.

Of the directly poisonous dusts, the most widely prevalent are those of certain lead compounds, particularly the oxide, carbonate, and the chromate. The dust is readily absorbed by the mucous membrane; some dust passes into the stomach and is dissolved by the gastric juice (*6*).

According to Fairhall, perforated nasal septum is a common occurrence among workers with bichromate dusts (*4*). In a study made by the United States Public Health Service it was found that continuous daily exposure to concentrations of chromic acid mist greater than 1 milligram in 10 cubic meters is likely to cause definite injury to the nasal tissues (*28*). It is believed that a similar concentration of the dust would be equally toxic.

In the case of poisoning from some heavy metals, there may be exposure to both dust and vapor. For instance, investigations have shown the safe limit of *total* exposure to lead oxide dust and fumes to be less than 1.5 mg per 10 cubic meters of air, except for prolonged exposure (*29*). In exposure to mercury dust and vapor, it was shown that the incidence of chronic mercurialism increased rapidly with increasing mercury concentration, after such concentration exceeds 2.0 mg per 10 cubic meters (*30*).

Alkalis and metallic oxides are common causes of dermatoses. Lye, potash, and lime are known to cause irritation to plasterers, cement makers, bricklayers, masons, stonecutters, modelers, and metal platers (*16*). Ulceration and perforation of the nasal septum occur among workers exposed to the dust of soda ash; systemic poisoning also occurs from inhalation of calcium cyanimide dust (*21*).

Cases of dermatitis, scleroderma, and cancer are reported to have been caused by exposure to dust of arsenic compounds. Certain aluminum salts are skin irritants, and aluminum dusts may contribute to the infection of skin and mucous membranes, through mechanical action (*16*).

(*b*) *Fibrosis-producing dusts.*—The most important of these are the inorganic, slightly soluble dusts which cause fibrous changes in the lung tissues, some of which are serious, and some of which cause little or no disability (*7*). So far as is known, no inorganic substances other than silicon derivatives cause more than a very moderate degree of fibrosis of the lung. Moreover, there seems to be no evidence that any other constituent of ordinary dusts can influence so unfavorably a pulmonary infection (*8*).

Although other dusts, when inhaled in sufficient concentrations over a long enough period of time, have been shown to be capable of producing a pulmonary fibrosis, nevertheless, the pneumoconiosis characterized by nodular fibrosis has to date been shown clinically and experimentally to be associated only with the inhalation of dusts containing free silica. Since this dust, to exert its harmful action, must enter the finer divisions of the lung, the particle size of the atmospheric dust may bear a definite relationship to the injurious effect produced. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The greater majority of particles found upon microscopic examination of the lung fall within the limits of from 1 to 3 microns (*31*). Examples of siliceous dusts are granite, quartz, sand, pumice, slate, and similar substances.

In a recent study among anthracite miners, the correlations between exposures to dust (which contained silica) and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed. Like correlations were found between the silica exposure and the extent of pulmonary changes (*32*). When the inhaled dust consists of silica combined with bases, silicates, some degree of change in the pulmonary tissue may result. In this respect asbestos dust seems to be unique among silicates in the prevalence and severity of the disease it causes (*33*).

The chief distinction between silicosis and conditions due to simple reactions caused by other dusts is the active proliferative reaction in the tissues which results in progressive nodulation. Silicosis, when

once established, strongly predisposes the lungs to infection, especially with the tubercle bacillus. Chronic interstitial pneumonia, chronic bronchitis, and emphysema are frequent complications of advanced degrees of silicosis.

The relation of the acute respiratory infections to the reaction due to dusts other than free silica has never been established, though a heightened incidence has often been shown statistically in workers in dusty trades. It is fairly certain that a dust-damaged lung, whatever the cause, fares much worse if an acute infection does supervene upon it (*7*).

In a study conducted by the Public Health Service among marble finishers in Vermont, it was found that marble dust, when inhaled in the concentrations found in the examined plants, produced a mild, bilateral, linear fibrosis in some cases, but no serious lung changes were noted and there was no disability due to the dust, even after years of exposure (*34*).

(*c*) *Non-fibrosis-producing dusts.*—These are inert, that is, they do not cause fibrous tissue to be produced, but may become encapsulated or lie free in the tissues, or they are absorbed without production of fibrous tissue. Included among them are alundum, coal, corundum, emery, limestone, magnesite, marble, plaster of paris (gypsum), and polisher's rouge.

DUST CONTROL

Engineering and medical control are the two most important factors in combating the industrial dust hazard, and are to a large extent complementary.

Engineering control.—As Lanza (*35*) has stated in a recent paper, "It is a basic principle in dealing with a dust hazard that the dust should be attacked at its point of origin and thus prevented from being disseminated into the atmosphere." After the dust has been spread throughout the air it is difficult to deal with it, and reliance must be placed on individual protection, which is never wholly satisfactory.

Lanza further cites various methods used in controlling dust. These will be reviewed but briefly here.

Dust may be entrapped at its source by suction devices and thus removed and collected. Familiar examples are exhaust hoods in grinding operations and the devices used in rock drilling. Generally speaking, the exhaust ventilation method, where applicable, is to be preferred in controlling a dust hazard. Water may be used to entrap dust and prevent its dispersal, and under certain circumstances it may be of advantage to combine the use of water and the use of suction. Sometimes a dusty process can be completely enclosed in a sealed room or compartment. It must be remembered, however, that

any mechanical device of this kind offers adequate protection only if it is properly designed, installed, and maintained.

A great deal of attention has been given the subject of individual protection from dust, and there are many types of respiratory protective devices now available. These are generally of two types—those which provide fresh air from an uncontaminated source and those which rely upon a filtering medium for removing dust from the air breathed. Where the use of such a device is indicated, only one of the types approved by the United States Bureau of Mines should be used. As a rule, it may be said that masks, respirators, or other such protective devices should be used only where exposure to the dust is intermittent and brief, or where some unusual condition makes a more adequate dust control impracticable (*35*).

Where bacteria or other living dusts in the air are associated with a process, sterilization methods, such as increased temperature, ultraviolet radiation, and chemicals like chlorine or other bactericidal substances, may be of use. Pasteurization temperature (about 140° F.) will kill most organisms except those bearing spores. Steam disinfection is used for horsehair, and proves to be practicable if the temperature does not exceed 230° F. Wool fibers, however, lose their elasticity by steam disinfection, and the "Duckering" process now used in England includes soaking of the wool in a formaldehyde solution, and drying in a current of air at a temperature of 160° F. (*12*). There are but few occupations in which there would be a sufficient concentration of dead bacteria to cause untoward effects in man.

In the case of dusts producing external irritation, auxiliary protective measures may include the use of protective clothing, gloves, goggles, and aprons, as well as protective salves, ointments, or other compounds to delay or diminish the irritant action. General rules for hygiene and good housekeeping should also be observed.

Medical control.—Equally important, and closely interrelated with the engineering phase, is medical control of occupational hazards. In addition to directing the proper placement of new workers and guarding the health of all employees, medical control is a check on the efficacy of the engineering control methods already instituted, or a measure of the need for new protective devices.

It has been stated that "Industry has found that the best way to treat industrial injuries and illness is to prevent them" (*36*), and medical control, through preemployment and periodic physical examinations, is one of the most important factors in such prevention.

The preemployment examination is made to determine the employee's physical and mental fitness for work. It serves to disclose

the presence of any contagious disease, reveals any minor physical defects which might later become serious, or whether the examinee's condition precludes his employment in certain or in all types of work. It should be remembered that such preemployment examinations are not to be made for the purpose of eliminating or excluding an employee, but rather for allocating him to the type of work for which he is physically suited. A worker should be given employment unless totally unfit, or unless his disability would cause him to be a hazard to himself or his associates. Furthermore, the practice of preemployment examinations should be extended to include executives and officials of industrial organizations.

"The purpose of periodic physical examinations is to secure and maintain physical fitness and thereby lengthen work spans" (*36*). Reexamination of employees sometimes results in the discovery of defects and disabilities which were not observed at the time of employment. In such cases an occupational adjustment should be made to provide continued employment, and remove the risk of permanent injury. Reexamination of employees is required by law in certain occupations in which the handling of poisonous or otherwise deleterious substances may result in the contraction of disease (*37*). Since some occupational diseases tend to clear up and recur, records of previous occupations should be included in the physical examinations. The frequency of examinations should be determined by the medical director, unless otherwise specified by law. Those exposed to known occupational disease hazards may have weekly or monthly examinations (*36*).

"It should always be kept in mind that the basic principle of physical examinations in industry is to keep men on the job and not allow the physical examination to be merely a weeding out process" (*35*).

REFERENCES

(*1*) Bloomfield, J. J., and Dalla Valle, J. M.: The determination and control of industrial dust. Public Health Bulletin No. 217. U. S. Govt. Printing Office, Wash., D. C., April 1935.
(*2*) Collis, Edgar L.: Industrial pneumoconioses, with special reference to dust phthises. Milroy Lectures, 1915. H. M. S. O., London.
(*3*) Sollmann, Torald: A manual of pharmacology. W. B. Saunders & Co., Philadelphia, 1932.
(*4*) Fairhall, Lawrence T.: Toxic dusts and fumes. J. Ind. Hyg. and Toxicol., November 1936.
(*5*) Bloomfield, J. J.: The sampling and analysis of industrial dusts. American Public Health Association Yearbook, 1935–36.
(*6*) Gibbs, W. E.: Dust hazard in industry. Ernest Benn, Ltd. London, 1925.
(*7*) Air Hygiene Foundation of America, Inc. Silicosis and allied disorders. Medical series, Bulletin No. 1. Pittsburgh, Pa., April 15, 1936.
(*8*) Kettle, E. H.: The action of harmful dusts. Inst. of Mining and Metallurgy, London, June 14, 1934.

(9) Dusts, fumes, and smokes. Occupation and health series, International Labour Office, Geneva, 1930.
(10) Drinker, Philip: Causation of pneumoconiosis. J. Ind. Hyg. and Toxicol., October 1936.
(11) Dyeing. Occupation and health series, International Labour Office, Geneva, 1930.
(12) Legge, Sir Thomas: Industrial maladies. Oxford University Press, London, 1934.
(13) Burstein, A.: Nicotine action from inhaled tobacco dust. J. Ind. Hyg., December 1927.
(14) White, R. Prosser: The dermatergoses, or occupational affections of the skin. H. K. Lewis & Co. Ltd., London, 1934.
(15) Schwartz, Louis: Skin hazards in American industry. Public Health Bulletin No. 215. Govt. Printing Office, Wash., D. C., October 1934.
(16) Downing, J. G.: Industrial dermatoses: Treatment and legal aspects: Review of recent literature. J. Ind. Hyg. and Toxicol., July 1935.
(17) Occupational lesions in workers in sugar. Belgium Letter, J. Am. Med. Assoc., Sept. 10, 1927. Abst. *in* J. Ind. Hyg., February 1928.
(18) Flour mills. Occupation and health series, International Labour Office, Geneva, 1930.
(19) Mayers, May R.: Susceptibility to dermatitis. Ind. Bull., Albany, N. Y., Vol. 16, No. 2, February 1937.
(20) Drinker, Philip, and Hatch, Theodore. Industrial dust. McGraw-Hill Book Company, Inc., New York, 1936.
(21) Schwartz, Louis: Skin hazards in American industry. Part II. Public Health Bulletin No. 229. Govt. Printing Office, Wash., D. C., September 1936.
(22) Poisonous woods. Occupation and health series, International Labour Office, Geneva, 1930.
(23) Hope, E. W., Hanna, W., and Stallybrass, C. O.: Industrial medicine and hygiene. Bailliere, Tindall, and Cox, London, 1923.
(24) Brown, G. T.: The diagnosis of bacterial allergy. Southern Med. J., Vol. 27, No. 10, October 1934.
(25) Sayers, R. R., and Meriwether, F. V.: Miliary lung disease due to unknown cause. Pub. Health Rep., Dec. 5, 1930.
(26) Actinomycosis. Occupation and health series, International Labour Office, Geneva, 1930.
(27) Holland, J. W.: Textbook of medical chemistry and toxicology. W. B. Saunders Company, Philadelphia, 1920.
(28) Bloomfield, J. J., and Blum, Wm.: Health hazards in chromium plating. Pub. Health Rep., Sept. 7, 1928.
(29) Russell, Jones, Bloomfield, Britten, and Thompson: Lead poisoning in a storage battery plant. Pub. Health Bull. No. 205. Govt. Printing Office, Wash., D. C., June 1933.
(30) Neal, Jones, Bloomfield, DallaValle, and Edwards: A study of chronic mercurialism in the hatters' fur cutting industry. Pub. Health Bull. No. 234. Govt. Printing Office, Wash., D. C., May 1937.
(31) Sayers, R. R., and Jones, R. R.: Silicosis and similar dust diseases. National Silicosis Conference, Washington, D. C., Apr. 14, 1936.
(32) Sayers, Bloomfield, DallaValle, Jones, Dreessen, Brundage, and Britten: Anthraco-silicosis among hard coal miners. Pub. Health Bull. No. 221. Govt. Printing Office, Wash., D. C., December 1935.
(33) Middleton, E. L.: Industrial pulmonary disease due to the inhalation of dust. The Lancet, July 4 and 11, 1936.
(34) Dreessen, Waldemar C.: Effect of inhaled marble dust as observed in Vermont marble finishers. Pub. Health Rep., June 22, 1934.
(35) Lanza, A. J.: Control and prevention of silicosis. Symposium on silicosis. California Tuberculosis Association, San Francisco, 1937.
(36) Newquist, M. N.: Medical service in industry and workmen's compensation laws. American College of Surgeons, Chicago, 1934.
(37) Medical care of industrial workers. National Industrial Conference Board, Inc. New York, 1926.

THE PRODUCTION OF TUMORS IN MICE OF STRAINS C_3H AND Y BY DIBENZANTHRACENE AND METHYLCHOLANTHRENE[1]

By H. B. ANDERVONT, *Biologist, United States Public Health Service*

It has been shown (*1*) that subcutaneous injections of a lard solution of 1:2:5:6-dibenzanthracene induces sarcomas at the site of injection in pure-strain mice. In this laboratory mice of various inbred strains have been given subcutaneous injections of lard-dibenzanthracene solutions and, until the experiments recorded in this paper, mice of strain *CBA* have proved to be the most resistant (*2*) of all strains. Boyland and Warren (*5*) found strain *CBA* mice to be less susceptible to the carcinogenic action of methylcholanthrene than the "Simpson Albino strain" of mice. The object of this brief report is to record experimental evidence showing that mice of strain *Y* are also very resistant to the carcinogenic action of both 1:2:5:6-dibenzanthracene and methylcholanthrene.

EXPERIMENTAL ANIMALS

Mice of two strains were used, all of which were bred in these laboratories. The establishment of the C_3H strain has been recorded by Strong (*7*) and their susceptibility to spontaneous growths, as observed in this laboratory, has been reported (*3*) previously. Of the pure-strain mice which have been injected subcutaneously with 1:2:5:6-dibenzanthracene in this laboratory, mice of strain C_3H have, thus far, responded to the injections by developing tumors earlier than any other strain.

Mice of strain *Y* were the offspring from a litter procured from the Roscoe B. Jackson Memorial Laboratories and the strain has been described by Dr. C. C. Little (*6*) of that laboratory. The mice are of yellow, brown, or black coat color and have been inbred by brother-to-sister mating for approximately 10 years. They have a low incidence of spontaneous mammary gland tumors and a medium incidence of "internal tumors."

EXPERIMENTAL

The first indication of the resistance of strain *Y* mice to the carcinogenic power of 1:2:5:6-dibenzanthracene was from the results obtained by injecting mice of strain *Y* and strain C_3H in two different experiments. Both strains, however, were injected with the same lard solution of the hydrocarbon. The experiment is described below:

---

[1] From the Office of Cancer Investigations, U. S. Public Health Service, Harvard Medical School, Boston, Mass.

Experiment 1.—On November 1, 1935, 25 strain *Y* mice (13 females and 12 males), consisting of 17 yellow, 5 brown, and 3 black animals, were each injected subcutaneously in the right axilla with 0.2 cc of a lard-solution of 1:2:5:6-dibenzanthracene containing 4 mg of the hydrocarbon dissolved in each cc of lard. The injections were repeated on November 15, 1935. Each animal thus received 1.6 mg of the compound dissolved in 0.4 cc of lard.

On November 15, 1935, each of 36 strain C_3H male mice received a single subcutaneous injection in the right axillary region, consisting of 0.8 mg of the compound dissolved in 0.2 cc of lard. The use of these mice has been recorded as experiment 4 in an earlier communication (*4*).

It will be noted that the strain *Y* mice received twice the amount of the carcinogenic agent that the strain C_3H animals received, and, as stated before, the same lard solution of the hydrocarbon was used to inject all mice of both strains. The results of the experiments are presented in table 1, where it is seen that the mice of strain C_3H developed tumors earlier than did those of strain *Y*. The average time of appearance of tumors in strain C_3H mice was 19.2 weeks, while for mice of strain *Y* it was 26.1 weeks.

Experiment 2.—In this experiment mice of both strains were tested for their susceptibility to lard solutions of 1:2:5:6-dibenzanthracene and methylcholanthrene. Lard solutions containing 4 mg of hydrocarbon in each cc were prepared by Dr. M. J. Shear, who also supplied the methylcholanthrene for the experiment. On December 23, 1936, 37 strain C_3H mice and 35 black strain *Y* mice each received a single subcutaneous injection in the right axilla of 0.2 cc of a lard solution containing 0.8 mg of one of the hydrocarbons. Of these, 17 strain C_3H males and 17 strain *Y* mice (10 males and 7 females) were injected with the lard solution of 1:2:5:6-dibenzanthracene while 20 strain C_3H males and 18 strain *Y* females were given the lard solution of methylcholanthrene.

The first tumors were noted in methylcholanthrene-injected strain C_3H mice on February 17, 1937, just 8 weeks after injection. The first tumor resulting from the injection of 1:2:5:6-dibenzanthracene was observed in a strain C_3H mouse on March 3, 1937, or 12 weeks after injection. So far as the *Y* mice were concerned, the first tumor was noted in a methylcholanthrene-injected mouse on April 8, 1937, or 16 weeks after injection, while of the dibenzanthracene-injected animals, none had developed a tumor 9 months after injection. The time of appearance of tumors is recorded in table 1.

TABLE 1.—*Experiments 1 and 2: Time of appearance of induced tumors in mice of strains C_3H and Y following subcutaneous injection of lard solutions of 1:2:5:6-dibenzanthracene or methylcholanthrene*

Experiment No.	Strain of mice	Hydrocarbon injected	Amount of hydrocarbon injected	Number of mice injected	Time in weeks: 8	10	12	14	16	18	20	22	24	26	28	30	32	34	Total number of tumors	Number dying without tumor	Number living and tumor-free on September 1, 1937
					Number of tumors																
			Mg																		
1	*Y*	Dibenzanthracene.	1.6	25	---	---	---	1	---	1	---	1	5	4	5	---	3	1	21	4	0
1	C_3H	do	.8	36	---	---	---	5	6	6	7	6	3	---	2	---	---	---	35	1	0
2	*Y*	do	.8	17	---	---	---	---	---	---	---	---	---	---	---	---	---	---	0	8	9
2	C_3H	do	.8	17	---	---	1	---	2	3	4	2	1	1	1	---	---	---	15	2	0
2	*Y*	Methylcholanthrene.	.8	18	---	---	---	---	2	---	2	1	1	---	2	---	1	1	10	2	6
2	C_3H	do	.8	20	11	4	1	3	---	---	---	---	---	---	---	---	---	---	19	1	0

The findings in the two experiments reveal clearly that mice of strain *Y* are more resistant than those of strain C_3H to the carcinogenic power of both hydrocarbons. Attention is directed to the fact that 0.8 mg of 1:2:5:6-dibenzanthracene failed to evoke tumors in strain *Y* mice within 36 weeks after injection, while the average time of appearance of tumors in strain C_3H mice after injection was 20 weeks. It should be mentioned that of the 8 mice of strain *Y* which died following the injection of 1:2:5:6-dibenzanthracene, 7 died prior to March 3, 1937, at which time the first tumor was noted in a strain C_3H animal. These 7 mice were males and died from wounds caused by fighting. Of the 10 remaining strain *Y* mice, one died, tumor-free, 30 weeks after injection and the other 9 were living and free from tumor on September 1, 1937.

The results attending injection of methylcholanthrene also revealed a difference in susceptibility between the strains, for the average time in which tumors appeared in the C_3H mice was 9.6 weeks, while for the *Y* mice, it was 24 weeks. Furthermore, six strain *Y* mice were living and were without tumor on September 1, 1937.

Little (*6*) found that in hybrid mice derived by crossing strains *Y* and *D* (dilute brown) the yellow animals had a lower incidence of mammary tumors than did the nonyellows. The results of the experiments recorded herein fail to give any information as regards the relation of coat color of strain *Y* mice to susceptibility to induced tumors, for most of the mice of experiment 1 were of yellow coat color and received 1.6 mg of 1:2:5:6-dibenzanthracene, while all the mice of experiment 2 were black and received 0.8 mg of the carcinogenic compounds. The results obtained in the yellow strain *Y* mice of experiment 1 failed to reveal any influence exerted by sex upon the appearance of induced subcutaneous growths.

So far as strain C_3H mice are concerned it is shown that they respond very early to the cancer-inducing power of methylcholanthrene, which is known to be highly carcinogenic, for 0.8 mg of the compound induced tumors in 75 percent of them within 10 weeks after injection, which indicates that these mice are very susceptible to carcinogenic hydrocarbons other than 1:2:5:6-dibenzanthracene.

SUMMARY

Mice of strains Y and C_3H have been tested for susceptibility to lard solutions of 1:2:5:6-dibenzanthracene and methylcholanthrene by subcutaneous injections. It was found that mice of strain Y are far more resistant than those of strain C_3H to the carcinogenic power of both hydrocarbons.

REFERENCES

(1) Andervont, H. B.: The production of dibenzanthracene tumors in pure strain mice. Pub. Health Rep., **49**: 620 (1934).
(2) ———— Further studies on the production of dibenzanthracene tumors in pure strain and stock mice. Pub. Health Rep., **50**: 1211 (1935).
(3) Andervont, H. B., and McEleney, W. J.: Incidence of spontaneous tumors in a colony of strain C_3H mice. Pub. Health Rep., **52**: 772 (1937).
(4) Andervont, H. B., and Lorenz, E.: Dibenzanthracene tumors in mice. The production of subcutaneous, pulmonary, and liver tumors by serum dispersions and lard solutions. Pub. Health Rep., **52**: 637 (1937).
(5) Boyland, E., and Warren, F. L.: The induction of tumors by methylcholanthrene in two strains of mice. J. Path. and Bact., **45**: 171 (1937).
(6) Little, C. C.: The relation of coat color to the spontaneous incidence of mammary tumors in mice. J. Exp. Med., **59**: 229 (1934).
(7) Strong, L. C.: The establishment of the C_3H inbred strain of mice for the study of spontaneous carcinoma of the mammary gland. Genetics, **20**: 586 (1935).

PULMONARY TUMORS IN MICE

V. FURTHER STUDIES ON THE INFLUENCE OF HEREDITY UPON SPONTANEOUS AND INDUCED LUNG TUMORS[1]

By H. B. ANDERVONT, *Biologist, United States Public Health Service*

In an earlier publication (*1*) experimental evidence was presented which showed that the susceptibility of the lungs of certain mice to the formation of tumors induced by subcutaneous injections of a lard-dibenzanthracene solution is inherited as a dominant character. Reciprocal cross-breeding was carried out between a strain of mice exhibiting a high incidence of spontaneous pulmonary tumors (strain *A*) and a strain in which very few such growths appear (strain *C* 57 black), and the progeny of this mating were designated as of the first

[1] From the Office of Cancer Investigations, U. S. Public Health Service, Harvard Medical School, Boston, Mass.

hybrid generation. Females of the first hybrid generation were then mated to male litter mates to procure animals designated as of the second hybrid generation. When the hybrid mice were injected subcutaneously with a lard-dibenzanthracene solution, 90 percent of the first generation and 74.7 percent of the second generation developed lung nodules.

Further studies on the inheritance of susceptibility to both spontaneous and induced pulmonary neoplasms in mice are reported in this paper. There are also included the results of experiments in which lard solutions of 1:2:5:6-dibenzanthracene were removed from injected mice and reinjected into other mice. This latter work is recorded herein because mice of the second hybrid generation were used as a source of material to be reinjected. This report is, therefore, divided into three sections, as follows: (1) The influence of heredity upon the development of spontaneous lung tumors in out-cross mice; (2) the influence of heredity upon the development of induced lung tumors in back-cross mice; (3) the ability of a lard-dibenzanthracene solution to induce tumors after previous injection into other mice.

THE INFLUENCE OF HEREDITY UPON THE DEVELOPMENT OF SPONTANEOUS LUNG TUMORS IN OUT-CROSS MICE

This part of the paper may be regarded as a continuation of the earlier publication (*1*) in which hybrid mice were injected subcutaneously with the carcinogenic compound dissolved in lard. The animals included herein were set aside as normal uninjected controls at that time. As stated previously, mice of the first hybrid generation were procured by mating mice of strains *A* and *C* 57 black on July 15, 1935, and there were 179 black offspring born between August 15 and October 5, 1935. Most of these mice were injected with the carcinogenic compound, but 30 females, born between August 15, 1935, and September 15, 1935, were set aside as normal controls. Four of them died prior to March 1, 1937, and were not autopsied because they had been eaten by other mice. Of the 26 remaining mice, 11 were sacrificed on July 23, 1936, when they were approximately 11.5 months old; 10 of them were tumor-free and 1 had a single pulmonary growth. The remaining 15 mice were kept until March 1, 1937, when they were approximately 18 months of age, and then killed and their lungs examined carefully for the presence of macroscopic tumor growth. Fourteen had pulmonary tumors and 1 was negative.[2]

Mice of the second hybrid generation were derived by mating females of the first hybrid generation to male litter mates on October 30, 1935. There were 665 animals obtained from this mating, and,

[2] It has been observed that the majority of strain *A* mice show cystic degeneration of the kidneys when 18 months of age. None of the first hybrid generation mice of this experiment showed similar lesions.

of these, 89 which were born between November 20, 1935, and December 15, 1935, were set aside as normal controls. The color and sex of the control mice of the second hybrid generation were divided as follows:

	Female	Male	Total
Black	18	15	33
Albino	16	19	35
Brown	8	13	21
Total	42	47	89

Two of these mice died when 6.5 months of age; both were free from tumor growth. Twenty-six were killed on July 23, 1936, when they were approximately 8 months old; 25 were tumor-free and 1 had a single pulmonary growth. Nine died between July 23, 1936, and March 8, 1937; all were negative so far as lung tumors were concerned. The remaining 52 mice were all sacrificed on March 8, 1937, when they were about 16 months old; 20 were lung-tumor free and 32 had macroscopic lung nodules.

The findings in the hybrid generations confirm the observations of Lynch (*4, 5*), as well as those of Bittner and Little (*3*), who have shown that, in mice, susceptibility to spontaneous lung neoplasms is inherited according to genetic principles. This is of special interest in view of recent evidence (*3*) that spontaneous mammary cancer in mice may be caused by an extra-chromosomal influence.

THE INFLUENCE OF HEREDITY UPON THE DEVELOPMENT OF INDUCED LUNG TUMORS IN BACK-CROSS MICE

In order to procure back-cross animals, female mice of the first hybrid generation were mated to males of strain *A* or strain *C*57 black on June 24, 1936. Twenty-four progeny were obtained from mating hybrids back to the strain *A* parent stock; these are called strain *A* back-cross, or *ABC* mice. There were 42 offspring resulting from mating hybrids back to the strain *C* 57 black parent stock; these are designated as strain *C* 57 back-cross, or *BBC* mice.

All the 66 back-cross mice received a subcutaneous injection of a lard-dibenzanthracene solution in the right axillary region on October 9, 1936, each mouse receiving 1 mg of 1:2:5:6-dibenzanthracene in 0.25 cc of lard.

The first subcutaneous tumor was noted on January 12, 1937, approximately 14 weeks after injection. Between January 12, 1937, and March 19, 1937, 20 of the *ABC* and 26 of the *BBC* animals developed subcutaneous sarcomas at the site of injection. As these mice died or were killed their lungs were examined for the presence of macroscopic tumor growths. On March 19, 1937, there were 4 *ABC*

and 16 *BBC* mice alive and free from tumor at the site of injection. All these were sacrificed and their lungs examined for the presence of tumor; all 4 *ABC* mice had multiple lung tumors, while 8 of the *BBC* animals had from 1 to 6 tumors in their lungs and the remaining 8 were free from tumor. The results of the experiment are presented in table 1.

TABLE 1.—*Subcutaneous and lung tumors in strain A back-cross and strain C 57 black back-cross mice following subcutaneous injection of a lard-dibenzanthracene solution*

	Strain *A* back-cross	Strain *C* 57 black back-cross
No subcutaneous or lung tumors	0	8
Subcutaneous tumors only	0	15
Lung tumors only	4	8
Both subcutaneous and lung tumors	20	11
Total	24	42

In table 1 it is seen that all of the 24 *ABC* and 19, or 45 percent, of the *BBC* mice developed pulmonary growths. All the *ABC* mice had "multiple nodules" within their lungs, a term used in this laboratory to designate lungs with 15 or more macroscopic tumors upon their surfaces, while in the 19 *BBC* animals with lung tumors there was a total of 101 such growths, an average of 5.3 tumors for each set of positive lungs. From these findings it is concluded that the lungs of the *BBC* mice were more resistant than those of the *ABC* mice to the development of tumors induced by the lard solution of 1:2:5:6-dibenzanthracene. The results are in harmony with those of Lynch (*6*) who utilized tar painting to demonstrate the inheritance of lung tumor susceptibility in back-cross mice and found that it was inherited as a dominant character.

The appearance of induced lung growths in 45 percent of the *BBC* mice in the experiment recorded above suggests that but one dominant factor may be involved in the inheritance of susceptibility to induced tumor when mice of strains *A* and *C* 57 black are used as test animals.

THE ABILITY OF A LARD-DIBENZANTHRACENE SOLUTION TO INDUCE TUMORS AFTER PREVIOUS INJECTION INTO OTHER MICE

As stated before (*1*), mice of the second hybrid generation were given 0.8 mg of 1:2:5:6-dibenzanthracene, dissolved in 0.2 cc of lard, subcutaneously in the right axilla on January 22, 1936, and the injection was repeated on February 5, 1936. Thus, each mouse received 1.6 mg of 1:2:5:6-dibenzanthracene dissolved in 0.4 cc of lard. On July 23, 1936, 6 months after the first injection, 62 of these animals were killed, 43 of which were free from tumor at the site of

the subcutaneous injection. Because part of the injected material was present in the subcutaneous tissues of some of the animals which did not have macroscopic tumors, it was considered of interest to determine whether it retained its cancer-inducing power. Recovery of the material was accomplished by drawing it through a 20-gage needle attached to a 0.5 cc calibrated syringe in which the amount was measured.

Two experiments were performed. In the first, material withdrawn from a mouse of the hybrid generation was measured, and injected immediately into the subcutaneous tissues of the right axillary region of a strain C_3H mouse. Animals of strain C_3H were used because experiments in this laboratory (*2*) have shown them to be very susceptible to the carcinogenic activity of 1:2:5:6-dibenzanthracene and, in addition, they had not been used as a parent stock from which the hybrids had been derived. The use of a foreign strain of mice reduced the possibility of obtaining tumors because of the presence of tumor cells within the injected material, for geneticists have shown that, as a rule, a tumor arising within a member of a strain of mice of known genetic constitution will not grow when implanted into members of another strain. Material was procured from 9 of the second hybrid generation mice and used to inject 9 males of strain C_3H. The amounts injected varied from 0.05 to 0.1 cc.

Subcutaneous sarcomas arose at the site of injection in 7 of the strain C_3H mice, the first appearing within 4 months and the last within 8 months after injection. The 2 surviving mice were killed and found to be free from tumor 10 months after injection.

In the second experiment material was withdrawn from 6 of the hybrid mice on July 23, 1936, pooled, and heated at 100° C. for 5 minutes in order to kill any living cells which were present in the lard. After cooling, the material was used to inject 5 strain C_3H mice, each animal receiving 0.1 cc subcutaneously in the right axilla. Four of these mice developed subcutaneous sarcomas at the site of injection; the first was noted 6 months and the last 9 months after injection.

These results show that the lard solution retained its ability to evoke tumors in strain C_3H mice after it had remained in the bodies of the hybrid mice for a period of 6 months.

CONCLUSIONS

Out-cross mice, procured by reciprocal matings between strain *A* mice which have a high incidence of spontaneous pulmonary growths and strain *C* 57 black mice in which very few such growths appear, developed spontaneous pulmonary tumors in accordance with genetic principles.

Back-cross mice, obtained by mating hybrids back to their strain *A* and strain *C* 57 black parent stocks inherited a susceptibility to pulmonary tumors induced by subcutaneous injection of a lard-dibenzanthracene solution, but the strain *A* back-cross mice were more susceptible than the strain *C* 57 black back-cross mice.

A lard solution of 1:2:5:6-dibenzanthracene produced tumors in the subcutaneous tissues of strain C_3H mice after remaining within the bodies of hybrid mice for a period of 6 months.

REFERENCES

(*1*) Andervont, H. B.: Pub. Health Rep., **52:** 304 (1937).
(*2*) Andervont, H. B.: Pub. Health Rep., **50:** 1211 (1935).
(*3*) Bittner, J. J., and Little, C. C.: J. Heredity, **28:** 117 (1937).
(*4*) Lynch, C. J.: J. Exp. Med., **43:** 339 (1926).
(*5*) Lynch, C. J.: J. Exp. Med., **54:** 747 (1931).
(*6*) Lynch, C. J.: J. Exp. Med., **46:** 917 (1927).

DEATHS DURING WEEK ENDED JANUARY 22, 1938

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Jan. 22, 1938	Corresponding week, 1937
Data from 86 large cities of the United States:		
Total deaths	8,991	[1] 10,578
Average for 3 prior years	9,970	
Total deaths, first 3 weeks of year	27,519	33,250
Deaths under 1 year of age	523	[1] 626
Average for 3 prior years	609	
Deaths under 1 year of age, first 3 weeks of year	1,624	1,990
Data from industrial insurance companies:		
Policies in force	69,764,818	68,976,571
Number of death claims	14,031	16,701
Death claims per 1,000 policies in force, annual rate	10.5	12.6
Death claims per 1,000 policies, first 3 weeks of year, annual rate	9.7	11.7

[1] Data for 85 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables a zero (0) is to be interpreted to mean that no cases or deaths occurred, while leaders (----) indicate that cases or deaths may have occurred although none were reported.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 29, 1938, and Jan. 30, 1937

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937
New England States:								
Maine	----	3	2	374	143	50	0	0
New Hampshire	----	----	10	1	62	12	0	0
Vermont	----	----	----	----	264	----	0	0
Massachusetts	2	5	----	----	192	863	0	4
Rhode Island	----	----	----	9	----	124	0	1
Connecticut	2	1	6	984	10	347	1	0
Middle Atlantic States:								
New York	33	40	[1] 14	[1] 208	564	296	11	7
New Jersey	12	10	12	163	1,011	440	1	5
Pennsylvania	37	48	----	----	5,953	84	9	10
East North Central States:								
Ohio	41	21	----	731	1,574	65	3	12
Indiana	83	21	13	322	340	13	1	4
Illinois	41	32	35	226	3,915	23	5	7
Michigan	18	19	1	83	971	44	1	2
Wisconsin	3	2	44	1,227	944	19	0	0
West North Central States:								
Minnesota	3	5	4	14	9	34	0	1
Iowa	29	4	----	556	98	----	4	0
Missouri	19	20	145	2,000	933	4	2	4
North Dakota	3	----	3	225	18	----	0	2
South Dakota	3	2	----	216	----	2	0	0
Nebraska	----	----	4	78	1	2	1	1
Kansas	7	9	25	3,640	553	6	1	1
South Atlantic States:								
Delaware	1	1	----	----	11	97	0	0
Maryland [2]	17	17	47	471	26	338	3	4
District of Columbia	9	7	3	130	12	32	0	2
Virginia	12	45	----	----	398	180	5	4
West Virginia	10	2	38	236	286	12	7	3
North Carolina [3]	30	33	47	34	976	54	5	3
South Carolina [3]	5	9	711	827	159	44	1	1
Georgia [3]	16	13	----	600	310	----	2	3
Florida	29	17	13	40	102	7	4	5
East South Central States:								
Kentucky	5	9	46	359	473	51	10	9
Tennessee	10	15	185	653	525	0	3	5
Alabama [3]	23	27	362	466	215	6	8	1
Mississippi [3]	5	8	----	----	----	----	2	0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 29, 1938, and Jan. 30, 1937—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937
West South Central States:								
Arkansas	13	7	190	864	196	3	1	1
Louisiana [3]	10	9	22	235	3	4	4	0
Oklahoma [4]	21	9	217	505	13	32	2	2
Texas [3]	73	50	719	2,435	60	324	3	0
Mountain States:								
Montana		1		3,343	6	4	0	0
Idaho	4	2	6	230	3	73	1	0
Wyoming				30	9	1	0	0
Colorado	10	7			174	3	0	1
New Mexico	4	4	1	930	157	26	0	0
Arizona	4	1	130	1,154	2	191	0	1
Utah [3]	6	2		7	54	154	1	0
Pacific States:								
Washington	4	13	4	415	21	69	0	2
Oregon	3		53	2,187	9	7	1	1
California	31	30	144	9,893	174	50	1	11
Total	691	580	3,256	37,101	21,929	4,190	104	120
First 4 weeks of year	2,761	2,507	11,628	108,828	71,269	16,790	377	560

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid and paratyphoid fevers		Whooping cough
	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938
New England States:									
Maine	0	0	22	21	0	0	5	0	55
New Hampshire	0	0	13	5	0	0	0	0	3
Vermont	0	0	13	10	0	0	0	0	31
Massachusetts	0	0	286	249	0	0	2	0	141
Rhode Island	0	0	28	74	0	0	0	0	59
Connecticut	0	0	92	108	0	0	0	0	39
Middle Atlantic States:									
New York	1	0	677	788	0	3	7	5	462
New Jersey	1	1	139	172	0	0	0	3	187
Pennsylvania	1	1	569	650	0	0	6	7	284
East North Central States:									
Ohio	0	2	486	438	8	8	2	0	149
Indiana	0	0	195	193	42	2	1	0	33
Illinois	3	2	837	551	84	32	2	8	112
Michigan	0	1	560	666	4	0	1	3	195
Wisconsin	0	0	221	348	12	13	0	2	198
West North Central States:									
Minnesota	1	0	178	147	35	5	0	0	53
Iowa	0	0	224	191	46	24	2	1	47
Missouri	1	0	231	234	48	97	8	1	49
North Dakota	0	0	28	29	23	20	0	0	80
South Dakota	0	2	13	116	3	4	0	0	23
Nebraska	0	2	47	70	2	2	0	0	8
Kansas	0	0	250	291	25	11	0	1	99
South Atlantic States:									
Delaware	0	0	14	12	0	0	0	1	10
Maryland [3]	1	0	67	57	0	0	2	1	44
District of Columbia	0	0	15	16	0	0	0	0	5
Virginia	0	1	41	30	0	0	2	8	109
West Virginia	0	0	51	47	0	0	1	3	143
North Carolina [3]	1	0	62	47	1	2	6	9	419
South Carolina [3]	0	0	1	6	0	0	1	2	44
Georgia [3]	0	1	11	16	0	0	3	3	66
Florida	1	0	11	11	1	0	1	1	22

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 29, 1938, and Jan. 30, 1937—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid and paratyphoid fevers		Whooping cough
	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938	Week ended Jan. 30, 1937	Week ended Jan. 29, 1938
East South Central States:									
Kentucky	2	0	85	24	34	0	2	3	49
Tennessee	0	1	32	26	2	0	3	9	30
Alabama [3]	1	0	31	14	0	2	1	2	31
Mississippi [2]	1	0	4	4	4	0	2	2	----
West South Central States:									
Arkansas	1	2	9	7	12	2	3	3	41
Louisiana [3]	1	1	16	5	0	0	4	4	6
Oklahoma [4]	0	1	49	22	29	0	3	1	38
Texas [3]	3	2	136	108	30	2	13	9	136
Mountain States:									
Montana	0	0	43	35	7	7	0	0	24
Idaho	0	0	29	39	30	2	1	0	50
Wyoming	0	0	14	5	2	0	0	0	14
Colorado	0	0	33	28	4	0	0	0	12
New Mexico	0	1	12	18	0	0	3	0	36
Arizona	0	0	11	33	0	0	3	0	55
Utah [2]	0	0	83	23	1	5	0	0	46
Pacific States:									
Washington	2	0	99	80	45	10	1	1	124
Oregon	0	3	70	15	11	18	0	0	28
California	4	2	221	306	30	4	4	8	405
Total	26	26	6,359	6,385	575	275	95	101	4,294
First 4 weeks of year	85	97	23,787	23,666	2,409	1,144	464	493	15,918

[1] New York City only.
[2] Week ended earlier than Saturday.
[3] Typhus fever, week ended Jan. 29, 1938, 36 cases, as follows: North Carolina, 2; South Carolina, 2; Georgia, 10; Alabama, 8; Louisiana, 1; Texas, 13.
[4] Figures for 1937 are exclusive of Oklahoma City and Tulsa.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week:

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
July 1937										
New Hampshire	----	3	1	----	----	----	3	21	0	1
August 1937										
New Hampshire	----	1	----	----	----	----	10	2	0	1
September 1937										
Vermont	----	4	----	----	22	----	13	12	0	10
Wisconsin	5	20	118	----	145	----	157	209	1	20
December 1937										
Alabama	31	108	746	92	66	10	6	87	1	14
Hawaii Territory	----	4	18	----	38	----	3	----	0	6
Illinois	20	158	116	10	4,365	----	12	2,315	111	9
Kansas	7	47	43	----	237	----	1	791	44	6
Nevada	3	1	9	----	3	----	0	5	0	0
New Mexico	2	27	9	1	306	----	1	107	0	22
New York	35	134	----	10	558	----	5	1,818	0	36
North Dakota	1	6	33	----	74	----	0	113	51	0
Texas	18	201	1,714	768	257	82	18	422	15	97
Wisconsin	1	11	141	----	523	----	1	610	17	2

Summary of monthly reports from States—Continued

	Cases
July 1937	
New Hampshire:	
Paratyphoid fever	1
September 1937	
Vermont:	
Chickenpox	14
German measles	1
Mumps	172
Undulant fever	2
Whooping cough	72
Wisconsin:	
Chickenpox	217
Dysentery (amoebic)	1
Encephalitis, epidemic or lethargic	2
German measles	52
Mumps	111
Septic sore throat	14
Tularaemia	1
Undulant fever	9
Whooping cough	967
December 1937	
Actinomycosis:	
Illinois	1
Chickenpox:	
Alabama	134
Hawaii Territory	35
Illinois	2,176
Kansas	997
Nevada	32
New Mexico	104
New York	2,657
North Dakota	139
Texas	341
Wisconsin	1,624
Dengue:	
Texas	41
Dysentery:	
Alabama (amoebic)	1
Illinois (amoebic)	5
Illinois (amoebic carriers)	13
Illinois (bacillary)	31
Kansas (bacillary)	1
New Mexico	2
New York (amoebic)	8
New York (bacillary)	97
Texas (bacillary)	43

	Cases
***December 1937*—Con.**	
Encephalitis, epidemic or lethargic:	
Alabama	2
Hawaii Territory	1
Illinois	6
Kansas	2
New York	9
Texas	1
Wisconsin	2
German measles:	
Alabama	2
Illinois	75
Kansas	13
New Mexico	1
New York	76
Wisconsin	35
Hookworm disease:	
Hawaii Territory	14
Impetigo contagiosa:	
Hawaii Territory	19
Illinois	14
Jaundice, infectious:	
Hawaii Territory	4
Mumps:	
Alabama	41
Hawaii Territory	7
Illinois	492
Kansas	562
Nevada	176
New Mexico	8
North Dakota	11
Texas	72
Wisconsin	330
Ophthalmia neonatorum:	
New York	7
Paratyphoid fever:	
Hawaii Territory	1
New Mexico	1
New York	5
Texas	2
Puerperal septicemia:	
Nevada	1
New Mexico	1
Rabies in animals:	
Alabama	72
Illinois	28
New Mexico	1
Rabies in man:	
Alabama	1
Illinois	1
Septic sore throat:	
Hawaii Territory	1
Illinois	6

	Cases
***December 1937*—Con.**	
Septic sore throat—Con.	
Kansas	6
New Mexico	7
New York	80
North Dakota	1
Wisconsin	18
Tetanus:	
Alabama	5
Hawaii Territory	2
Illinois	8
Kansas	1
New York	6
Trachoma:	
Alabama	2
Hawaii Territory	3
Illinois	50
Trichinosis:	
Hawaii Territory	4
New York	3
Tularaemia:	
Illinois	39
Kansas	9
New Mexico	2
Wisconsin	1
Typhus fever:	
Alabama	27
Hawaii Territory	5
Illinois	1
Texas	20
Undulant fever:	
Alabama	2
Hawaii Territory	1
Illinois	12
Kansas	18
New York	19
Texas	11
Wisconsin	5
Vincent's infection:	
Illinois	23
Kansas	13
New York	68
Whooping cough:	
Alabama	107
Hawaii Territory	25
Illinois	334
Kansas	310
Nevada	4
New Mexico	93
New York	1,493
North Dakota	79
Texas	582
Wisconsin	506

WEEKLY REPORTS FROM CITIES

City reports for week ended Jan. 22, 1938

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	232	1,780	207	2,434	1,114	1,865	27	398	21	1,145	----
Current week	163	221	73	5,258	800	1,648	46	365	20	1,040	----
Maine:											
Portland	1	----	0	1	6	1	0	0	0	31	27
New Hampshire:											
Concord	0	----	0	8	3	0	0	0	0	2	16
Manchester	0	----	0	0	4	8	0	0	0	0	14
Nashua	0	----	0	0	2	0	0	1	0	8	17
Vermont:											
Barre	0	----	0	19	0	0	0	0	0	0	1
Burlington	0	----	0	1	0	0	0	0	0	1	10
Rutland	0	----	0	0	2	0	0	0	0	0	18
Massachusetts:											
Boston	0	----	2	92	12	82	0	10	0	9	207
Fall River	0	----	0	0	5	6	0	0	0	21	37
Springfield	0	----	0	1	2	2	0	0	0	4	42
Worcester	0	----	0	0	7	9	0	4	0	8	54
Rhode Island:											
Pawtucket	0	----	0	0	0	3	0	0	0	0	16
Providence	0	----	0	0	7	31	0	6	0	13	72
Connecticut:											
Bridgeport	0	----	0	0	3	17	0	0	0	1	40
Hartford	0	4	0	0	5	12	0	0	0	5	38
New Haven	0	1	0	0	2	0	0	0	0	3	32
New York:											
Buffalo	0	----	0	2	13	32	0	8	0	12	150
New York	19	19	5	148	128	229	0	83	4	143	1,603
Rochester	0	----	0	2	7	5	0	1	0	11	74
Syracuse [1]	0	----	0	6	5	5	[1]0	0	0	7	53
New Jersey:											
Camden	1	----	1	45	0	9	0	1	0	0	35
Newark	3	1	0	4	10	17	0	6	0	17	100
Trenton	0	----	1	18	5	3	0	3	0	0	37
Pennsylvania:											
Philadelphia	5	4	2	274	38	109	0	21	1	35	530
Pittsburgh	2	----	5	477	30	51	0	8	1	18	197
Reading	0	----	0	3	2	7	0	1	0	1	45
Scranton	0	----	----	52	----	5	0	----	0	3	----
Ohio:											
Cincinnati	4	----	2	1	16	23	0	11	0	5	150
Cleveland	2	27	3	105	18	54	0	11	0	39	227
Columbus	1	1	1	81	9	5	0	2	0	1	85
Toledo	0	----	0	82	7	3	0	5	0	7	78
Indiana:											
Anderson	0	----	0	1	2	15	4	0	0	3	11
Fort Wayne	0	----	0	30	5	7	0	1	0	0	43
Indianapolis	41	----	3	54	23	28	0	5	0	6	121
South Bend	0	----	0	1	4	3	0	0	0	0	21
Terre Haute	5	----	0	9	0	1	0	0	0	0	28
Illinois:											
Alton	1	----	0	1	4	11	0	0	0	0	13
Chicago	8	22	1	1,328	71	240	0	32	1	45	730
Elgin	0	----	0	3	1	24	0	0	0	4	11
Moline	1	----	0	73	4	12	0	1	0	0	16
Springfield	1	----	0	0	7	4	3	0	0	0	22
Michigan:											
Detroit	9	----	2	531	19	178	1	12	0	83	186
Flint	1	----	1	1	3	41	0	1	0	21	27
Grand Rapids	0	----	0	4	4	29	0	0	0	5	38
Wisconsin:											
Kenosha	0	----	0	2	0	0	0	0	0	0	4
Madison	0	----	0	0	0	2	0	0	0	3	19
Milwaukee	0	1	1	728	10	22	0	2	0	15	112
Racine	0	----	0	2	1	6	0	0	0	0	10
Superior	0	----	0	0	0	2	1	1	0	1	12

[1] The report from the city health officer of 1 case of smallpox in Syracuse for the week ended January 1, 1938, Pub. Health Rep., January 21, p. 106, was an error, later information stating that no case had occurred.

City reports for week ended Jan. 22, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	------	1	0	4	4	1	0	0	6	22
Minneapolis	0	------	2	2	12	21	0	1	0	3	117
St. Paul	0	------	0	5	0	5	26	0	0	2	62
Iowa:											
Cedar Rapids	0	------	--------	1	--------	6	0	-------	0	5	--------
Davenport	0	------	--------	45	-------	3	0	-------	0	0	-------
Des Moines	0	------	--------	1	-------	21	2	-------	0	1	33
Sioux City	1	------	--------	0	-------	0	0	-------	0	0	--------
Waterloo	0	------	--------	0	-------	5	0	-------	0	0	--------
Missouri:											
Kansas City	1	------	0	92	22	13	0	0	0	3	123
St. Joseph	0	------	0	1	8	5	0	0	0	0	32
St. Louis	8	------	0	240	13	70	5	7	0	3	249
North Dakota:											
Fargo	0	------	0	0	1	7	0	0	0	0	13
Grand Forks	0	------	--------	0	-------	3	0	-------	0	0	--------
Minot	0	------	0	0	0	0	6	0	0	16	4
South Dakota:											
Aberdeen	0	------	--------	2	-------	0	0	-------	0	3	--------
Sioux Falls	0	------	0	0	0	0	0	0	0	0	9
Nebraska:											
Lincoln	0	------	--------	1	-------	6	0	-------	0	0	--------
Omaha	0	------	0	2	6	4	0	0	0	0	52
Kansas:											
Lawrence	0	------	0	2	1	0	0	0	0	2	9
Topeka	0	------	0	0	2	4	0	0	0	26	22
Wichita	0	------	0	1	2	3	0	1	0	2	33
Delaware:											
Wilmington	0	------	0	2	6	7	0	1	0	6	41
Maryland:											
Baltimore	4	9	4	3	25	28	0	9	2	35	236
Cumberland	0	------	0	0	1	0	0	0	0	0	15
Frederick	0	------	0	0	0	0	0	0	0	0	8
District of Columbia:											
Washington	9	3	2	12	20	15	0	8	3	3	160
Virginia:											
Lynchburg	2	------	0	1	1	0	0	0	1	6	15
Norfolk	0	------	0	101	6	16	0	2	0	2	29
Richmond	0	------	1	3	5	5	0	0	0	0	52
Roanoke	2	------	1	1	0	1	0	1	0	2	21
West Virginia:											
Charleston	0	1	0	35	4	1	0	0	0	2	30
Huntington	0	------	--------	33	-------	1	0	-------	0	0	--------
Wheeling	0	------	0	5	4	2	0	1	0	12	24
North Carolina:											
Gastonia	0	------	--------	0	-------	0	0	-------	0	0	--------
Raleigh	0	------	0	2	3	0	0	1	0	44	15
Wilmington	0	------	0	3	2	0	0	0	0	5	15
Winston-Salem	0	------	0	2	5	2	0	2	0	24	20
South Carolina:											
Charleston	1	44	3	9	4	3	0	3	0	0	28
Florence	0	------	0	0	0	1	0	1	0	0	10
Greenville	0	------	0	1	7	0	0	1	0	12	14
Georgia:											
Atlanta	1	15	1	175	8	7	0	0	0	12	102
Brunswick	0	------	0	0	0	0	0	0	0	0	2
Savannah	0	------	0	0	4	3	0	3	2	0	42
Florida:											
Miami	0	1	0	112	2	0	0	1	0	0	29
Tampa	1	1	1	1	2	1	0	0	0	0	23
Kentucky:											
Ashland	2	------	--------	3	-------	2	0	-------	0	12	--------
Covington	0	------	1	0	3	2	0	1	0	0	23
Lexington	0	------	0	0	3	0	0	1	0	0	18
Louisville	1	4	0	132	5	33	0	2	0	3	50
Tennessee:											
Knoxville	1	5	0	14	0	1	0	1	0	0	24
Memphis	1	2	2	209	5	4	0	7	0	5	88
Nashville	0	------	1	4	3	2	0	2	0	4	55
Alabama:											
Birmingham	2	21	4	38	7	5	0	3	0	0	74
Mobile	0	------	1	0	3	0	0	0	0	0	27
Montgomery	1	------	--------	4	-------	0	0	-------	0	5	--------

City reports for week ended Jan. 22, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Arkansas:											
Fort Smith	0	------	-------	0	-------	1	6	-------	0	2	--------
Little Rock	1	------	2	110	5	1	0	0	0	1	9
Louisiana:											
Lake Charles	0	------	0	0	1	1	0	1	0	1	13
New Orleans	3	8	5	0	20	8	0	15	4	1	187
Shreveport	0	------	0	2	6	4	0	2	0	0	41
Oklahoma:											
Oklahoma City	2	------	0	0	3	6	0	0	0	0	35
Tulsa	2	------	-------	1	-------	2	0	-------	0	25	--------
Texas:											
Dallas	3	7	4	0	10	11	0	4	0	0	78
Fort Worth	4	------	0	1	8	7	1	0	0	2	40
Galveston	0	------	0	0	2	1	0	1	1	0	19
Houston	5	------	1	1	8	5	0	5	0	0	93
San Antonio	2	------	2	0	11	0	0	12	0	0	72
Montana:											
Billings	0	------	0	0	1	0	0	0	0	0	4
Great Falls	0	------	0	0	3	0	0	0	0	23	8
Helena	0	------	0	0	0	1	0	0	0	0	3
Missoula	0	------	0	0	1	1	0	0	0	0	4
Idaho:											
Boise	0	------	0	0	0	0	8	0	0	8	4
Colorado:											
Colorado Springs	0	------	0	0	0	3	0	0	0	0	12
Denver	7	------	1	216	11	25	0	2	0	3	95
Pueblo	0	------	0	0	2	0	0	0	0	7	13
New Mexico:											
Albuquerque	0	------	0	18	2	2	0	1	0	4	6
Utah:											
Salt Lake City	0	------	1	3	2	20	0	1	0	6	40
Washington:											
Seattle	1	------	1	2	2	1	0	4	0	63	95
Spokane	0	------	0	1	4	4	0	0	0	9	36
Tacoma	0	------	0	0	5	7	1	0	0	22	22
Oregon:											
Portland	0	------	2	0	5	21	0	1	0	0	95
Salem	0	4	-------	1	-------	0	0	-------	0	0	--------
California:											
Los Angeles	6	25	2	7	30	52	0	17	0	31	371
Sacramento	0	------	0	0	3	4	0	1	0	33	26
San Francisco	0	------	0	1	12	11	0	12	0	66	183

State and city	Meningococcus meningitis		Poliomyelitis cases	State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Maine:				District of Columbia:			
Portland	1	0	0	Washington	1	1	0
New York:				Virginia:			
Buffalo	4	0	0	Lynchburg	1	0	0
New York	2	3	1	Alabama:			
Pennsylvania:				Birmingham	1	0	0
Philadelphia	2	1	0	Tennessee:			
Ohio:				Memphis	2	0	0
Cincinnati	0	0	1	Kentucky:			
Indiana:				Ashland	1	0	0
Indianapolis	0	0	1	Arkansas:			
Illinois:				Little Rock	0	1	0
Chicago	0	1	0	Louisiana:			
Minnesota:				New Orleans	0	1	0
Minneapolis	1	0	0	Shreveport	0	1	0
Missouri:				Texas:			
St. Louis	2	0	0	Dallas	1	0	0
Maryland:				California:			
Baltimore	2	0	0	Los Angeles	0	1	0

Encephalitis, epidemic or lethargic.—Cases: Newark, 1; New York, 2; Toledo, 1; Washington, D. C., 1.
Pellagra.—Cases: Winston-Salem, 1; Atlanta, 4; Birmingham, 2; New Orleans, 1; Dallas, 2; San Francisco, 1.
Typhus Fever.—Cases: Miami, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended January 15, 1938.—During the 2 weeks ended January 15, 1938, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis					1	1				2
Chickenpox		30	24	320	684	134	40	68	192	1,501
Diphtheria		4	5	100	11	4	1	7	1	133
Dysentery					1					1
Erysipelas				12	4	2	2	5	3	28
Influenza		3			40				37	80
Lethargic encephalitis									1	1
Measles		51	12	209	453	27	76	101	331	1,260
Mumps		6	1		159	52	10	4	56	288
Paratyphoid fever					1			1		2
Pneumonia		4			79		3		19	105
Poliomyelitis		1	2	4	2		1	3	1	14
Scarlet fever		17	19	257	258	73	109	66	32	831
Smallpox							1	1		2
Trachoma					1			1	1	3
Tuberculosis	2	13	15	92	93	2	3	2	29	251
Typhoid fever		1	5	51	6		1		5	69
Undulant fever					3			3		6
Whooping cough				230	96	30	11	7	95	469

CZECHOSLOVAKIA

Communicable diseases—October 1937.—During the month of October 1937, certain communicable diseases were reported in Czechoslovakia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	3		Paratyphoid fever	21	
Cerebrospinal meningitis	14	3	Poliomyelitis	41	4
Chickenpox	200		Puerperal fever	23	5
Diphtheria	4,260	196	Scarlet fever	2,793	26
Dysentery	974	110	Trachoma	79	
Influenza	37		Tularaemia	3	
Lethargic encephalitis	13	1	Typhoid fever	1,100	70
Malaria	249				

SWEDEN

Notifiable diseases—November 1937.—During the month of November 1937, cases of certain notifiable diseases were reported in Sweden as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	7	Poliomyelitis	[1] 209
Diphtheria	31	Scarlet fever	1,564
Dysentery	27	Syphilis	36
Epidemic encephalitis	1	Typhoid fever	5
Gonorrhea	1,026	Undulant fever	14
Paratyphoid fever	11	Weil's disease	1

[1] Includes 20 cases nonparalytic at time of notification.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for January 28, 1938, pages 144–159. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

Indochina (French).—During the week ended January 22, 1938, 7 cases of cholera were reported in Annam Province, and 2 cases of cholera in Tonkin Province, French Indochina.

Plague

Hawaii Territory—Island of Hawaii—Hamakua District—Paauhau Sector.—A rat found on January 17, 1938, and another on January 21, 1938, in Paauhau Sector, Hamakua District, Island of Hawaii, Hawaii Territory, have been proved plague infected.

Peru—Lima Department.—During the month of December 1938, 6 cases of plague with 5 deaths were reported in Lima Department, Peru.

×